Shock Pathophysiology

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Abstract: Shock, defined as the state where oxygen delivery to tissues is inadequate for the demand, is a common condition in veterinary patients and has a high mortality rate if left untreated. The key to a successful outcome for any patient in shock involves having a clear understanding of the pathophysiology and compensatory mechanisms associated with shock. This understanding allows more efficient identification of patients in shock based on clinical signs and timely initiation of appropriate therapies based on the type and stage of shock identified.

Shock is a condition that is commonly seen in practice but just as commonly is not completely understood. This review focuses on the body’s compensatory responses to shock and the clinical signs to help provide practitioners with a better understanding of what shock is and how it can be categorized. Treatment is discussed in the context of the pathophysiology but is not covered in depth.

Definitions
The first difficulty comes in defining shock. At its most elemental, the definition can be stated as:

\[ \text{oxygen delivery} \neq \text{oxygen consumption} \left( \text{DO}_{2} \neq \text{VO}_{2} \right) \].

Most cases of shock are the result of decreased delivery of blood to tissues. When blood is not delivered to tissues, oxygen is not delivered. Oxygen is critical for normal cellular function; when the tissues do not receive oxygen, normal cellular aerobic metabolism ceases and anaerobic metabolism ensues. As a result, cells are unable to produce adequate amounts of ATP (Figure 1) to sustain normal metabolic function, ultimately leading to cellular dysfunction and death. Additionally, sustained anaerobic metabolism results in the production of cytokines and substances such as lactate and nitric oxide, which further complicate shock (Figure 2).

Multiple factors determine oxygen delivery to cells (Figure 3); however, the simplest way to envision oxygen delivery is to consider the body’s cardiac output as being roughly equivalent to the blood delivered throughout the body. In turn, cardiac output is defined

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Figure 1. Aerobic versus anaerobic metabolism. TCA = tricarboxylic acid

Figure 2. Sequelae of prolonged anaerobic metabolism.

Figure 3. Circulatory collapse

Cellular dysfunction
Circulatory collapse
GI barrier breakdown + translocation of gut bacteria
Consumptive coagulopathy
Death
as heart rate times stroke volume. Appreciating the interrelationship between oxygen delivery and cardiac output is critical to understanding the pathophysiology of shock and guiding treatment.

It is less common that the body’s demand for oxygen is the driving force for the imbalance (i.e., that cardiac output is completely normal in a patient in shock). One example of this situation is overwhelming infection, in which the infection causes increased cellular metabolism (and therefore increased cellular oxygen demand). Increases in cellular metabolism alone can cause a state of shock before or in addition to the development of decreased cardiac output secondary to the infection.1,2

A second example in which cardiac output can be normal in a shock patient is when there is abnormal perfusion of tissues. When large numbers of cells are bypassed by oxygenated blood, an imbalance in oxygen demand and delivery develops that can lead to shock.1,3–8 In cases of abnormal perfusion, the microcirculation at the capillary and other small (≤100 μm) vessel level is typically affected.2,4,7 The microcirculation responds in a variety of ways, culminating in increased permeability of the walls of the endothelium and regions of vasodilation and altered blood flow.3 This

Figure 3. The determinants of oxygen delivery in the body. \(\text{CaO}_2 = \text{arterial oxygen content, } \text{CO} = \text{cardiac output, } \text{DO}_2 = \text{oxygen delivery, } [\text{Hb}] = \text{concentration of hemoglobin in the blood, } \text{HR} = \text{heart rate, } \text{PaO}_2 = \text{partial pressure of oxygen in arterial blood, } \text{SV} = \text{stroke volume, } \text{SpO}_2 = \% \text{hemoglobin saturation with oxygen.}\)

Table 1. Categories, Examples, Basic Definitions, and Pathophysiology of Shock

<table>
<thead>
<tr>
<th>Category of Shock</th>
<th>Classic Example</th>
<th>Basic Definition</th>
<th>Pathophysiology/Events Leading to Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absolute</td>
<td>Absolute: bleeding from wound (laceration)</td>
<td>Decreased effective circulating blood volume</td>
<td>Decreased effective circulating volume → decreased venous return → decreased stroke volume → decreased cardiac output and blood delivery to tissues</td>
</tr>
<tr>
<td>Relative</td>
<td>Relative: bleeding into third space in body (hemoabdomen, fracture hematoma)</td>
<td>Physical impediment to blood flow in large vessels (predominantly veins)</td>
<td>Physical blockage to venous return/blood trapped distal to obstruction → decreased stroke volume → decreased cardiac output and blood delivery to tissues</td>
</tr>
<tr>
<td>Obstructive</td>
<td>Gastric-dilatation volvulus (dilated stomach occludes caudal vena cava)</td>
<td>Heart unable to pump blood (typically caused by lack of contractility)</td>
<td>Decreased contractility → decreased cardiac output and blood delivery to tissues</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>Dilated cardiomyopathy</td>
<td>Multifactorial (one or more of the following):</td>
<td>Microcirculatory vasodilation → blood trapped in periphery → decreased venous return → decreased stroke volume → decreased cardiac output and blood delivery to tissues</td>
</tr>
<tr>
<td></td>
<td>Sepsis (Gram-negative endotoxemia)</td>
<td>1. Vasodilation, especially peripheral vessels (both microcirculation + macrocirculation)</td>
<td>Increased vessel wall permeability → decreased effective circulating blood volume → decreased venous return → decreased cardiac output and blood delivery to tissues</td>
</tr>
<tr>
<td></td>
<td>Anaphylaxis</td>
<td>2. Increased vessel permeability (relative hypovolemia as fluid leaks out of vessels)</td>
<td>Decreased cardiac contractility → decreased cardiac output and blood delivery to tissues</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Decreased cardiac contractility due to effects of cytokine mediators (sepsis) or platelet activating factor (anaphylaxis)</td>
<td>Multiple clot formation → small vessels occluded → decreased venous return → decreased cardiac output and blood delivery to tissues</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Activation of the coagulation system</td>
<td></td>
</tr>
</tbody>
</table>

leads to less blood being delivered to other cells and local hypoxia of the bypassed cells. Examples of such derangements in microcirculation include the systemic inflammatory response syndrome (SIRS) and reperfusion injury. Additionally, in human medicine, use of coronary artery bypass grafting can physically re-route blood away from tissues, causing those tissues to suffer from decreased oxygen delivery.

In an attempt to encompass and categorize the various types of shock, shock is typically divided into categories that help explain why oxygen delivery is not matching oxygen demand. However, it is important to remember that clinical cases of shock usually do not fall neatly into one category and often straddle several categories. The four categories described in this article are listed in Table 1, along with an explanation of why each category meets the basic definition of shock.

Compensation for Shock
Regardless of the cause, when tissues are not properly supplied with oxygen, the body attempts to remedy the situation by initiating a series of neural and hormonally mediated compensatory mechanisms. The end goal of these mechanisms is to increase cardiac output and blood vessel tone in an attempt to better supply the cells with oxygen. These compensatory mechanisms can be grouped into three separate categories: (1) effects exerted within minutes (acute), (2) effects exerted in 10 minutes to 1 hour (moderate), (3) effects exerted within 1 to 48 hours (chronic). In general, the body responds by increasing heart rate, increasing peripheral vascular tone, and attempting to increase stroke volume, all in an effort to improve cardiac output and keep perfusion to tissues intact. Stroke volume is improved by increasing the amount of blood returned to the heart (e.g., venous return). One way to increase venous return is to shunt blood from small (less important), peripheral vessels to the heart to supply the myocardium, lungs, and brain. The kidneys provide a second way to improve venous return by retaining fluid to bolster the total blood volume. Figure 4 summarizes these various compensatory mechanisms; the following text discusses the compensation in more detail.

Acute Compensatory Mechanisms

Catecholamines
Acute compensatory effects are limited to those affecting heart rate and redistributing peripheral blood back to the heart. They are mediated by the sympathetic nervous system (SNS) and catecholamine release and take effect within 30 seconds to a few minutes. As cardiac output decreases, impulse generation by the baroreceptors at the carotid sinus and aortic arch in the heart decreases. Under normal conditions, baroreceptor impulses work to inhibit the vasoconstrictor center of the medulla and increase stimulation of the vagal center in the brain, leading to vasodilation. When the baroreceptor impulses are decreased, the vasomotor center in the brain operates unchecked and SNS signals from the sympathetic nervous system (SNS) and cardiac output.

A second important stimulus of catecholamine secretion is hypoxemia. This can be true hypoxemia, represented by a global

Figure 4. Compensatory mechanisms in response to shock. ACE = angiotensin-converting enzyme, Ang = angiotensin, JG = juxtaglomerular, [Na] = concentration of sodium, SNS = sympathetic nervous system.
decrease in arterial oxygen content, or relative hypoxemia, caused by microcirculatory derangements that shunt blood away from tissues. Chemoreceptors that sense the oxygen content of the blood are located in the carotid artery and aorta. Those in the carotid artery sense decreased oxygen delivery to the brain and, therefore, stimulate the vasomotor center to increase SNS stimulation regardless of peripheral blood pressures. In the aorta, decreases in peripheral blood pressure are signaled by decreased baroreceptor stimulation and chemoreceptors are activated as a result of decreased oxygen delivery. Both baroreceptor and chemoreceptor signals lead to increased SNS signals from the vasomotor center in the brain.

Cortisol
Cortisol is also rapidly mobilized in the acute stages of shock (within minutes). Cortisol is released from the adrenal gland in response to corticotropin-releasing hormone (CRH) from the hypothalamus and also by stimulation via adrenocorticotropic hormone. Stimuli such as pain and mental or physical stress can lead to increases in CRH production. These stimuli are generated in or transmitted through the brain to the hypothalamus. Cortisol has many effects, and it is not completely understood which effect is the most important in shock; however, stimulation of glycogenolysis and mobilization of fat and protein stores for gluconeogenesis are often considered the most important. Release of glucose into the bloodstream provides a readily accessible energy source. We believe that the most important effects of this glucose surge are to supply endothelial cells in the blood vessels with energy to continue contraction, feed the myocardial cells to continue contraction, and allow brain cells to function in the short term.

Transcapillary Shifts
A final mechanism that aids in the acute improvement in blood volume is transcapillary shifting of fluid from the interstitium to the vasculature. This happens at the capillary level, primarily in cases of hypovolemic shock. When the pressure within the capillaries drops secondary to hypovolemia and vasconstrictive shunting of blood, Starling’s forces dictate that fluid will move from an area of higher pressure (the interstitium) into an area of lower pressure (the vessel). Fluid continues to move until the dilution of proteins in the blood vessels (decreasing oncotic pressure in the blood vessel) is balanced with the concentration of proteins in the interstitium (increasing oncotic pressure). Additionally, as fluid moves out of the interstitium into the vascular space, fluid volume and, therefore, pressure decrease in the interstitium (decreasing hydrostatic pressure).

An additional step during transcapillary fluid shifting involves movement of proteins into the blood from storage sites in the mesentery and liver. These proteins increase oncotic pressure in the blood vessels to continue to help draw fluid from the interstitium into blood vessels and maintain the extra fluid within the blood vessels.

Moderate Compensatory Mechanisms
The next level of compensation starts within about 10 minutes to 1 hour after the body enters the shock state.
**Angiotensin II**

Baroreceptors in the juxtaglomerular apparatus near the renal glomerulus sense decreased blood flow from decreased cardiac output. This decreases impulse generation in the baroreceptors, which in turn leads to renin secretion. Renin causes conversion of angiotensinogen to angiotensin I in the bloodstream. Angiotensin I is converted to angiotensin II in the lungs under the influence of angiotensin-converting enzyme. Angiotensin II binds to angiotensin receptors on the blood vessels and causes vasoconstriction.

The vasoconstriction not only improves blood vessel tone to maintain perfusion to the tissues but also, more importantly, forces blood from less important peripheral tissues (including the splanchnic circulation) to the brain and heart to improve venous return and cardiac output.

Angiotensin II also retains water and sodium in the kidneys to help maintain blood volume through renal artery vasoconstriction, which reduces filtration of blood through direct effects on the tubules that are not completely elucidated.¹

**Vasopressin**

Vasopressin is released from the posterior pituitary gland in response to increased osmolarity (i.e., less water and more sodium in the blood that passes by the osmoreceptors in the hypothalamus) or decreased effective circulating blood volume as sensed by the baroreceptors and stretch receptors (in the right and left atria). The atrial stretch receptors are active when there is a large volume in the atria and work to inhibit vasopressin secretion; when the atria are less full, more vasopressin is released because of lack of inhibition. Even small alterations—a 1% change in osmolarity or a 10% decrease in blood volume—lead to release of vasopressin.⁷ Other stimuli, including nausea and hypoxia, also develop in patients with shock and cause further release of vasopressin.⁷ Vasopressin binds to V1 receptors on the arterioles, causing vasoconstriction. As with angiotensin or norepinephrine, this improves vascular tone in an effort to maintain delivery of blood to tissues. Additionally, it increases return of blood from the peripheral tissues to the heart so that venous return and cardiac output are maintained.

**Chronic Compensatory Mechanisms**

If the patient survives the shock situation, the final stages of compensation involve replacing the blood volume in the body. This takes place from 1 to 48 hours after insult.

**Aldosterone**

At the same time that angiotensin II is exerting its effects on blood vessels and the kidney, it is also stimulating the adrenal glands to secrete aldosterone from the adrenal gland cortex.¹ Aldosterone

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### Table 2. Physical Examination Findings at Each Stage of Shock

<table>
<thead>
<tr>
<th>Physical Examination Finding</th>
<th>Compensated Shock</th>
<th>Acute Decompensated Shock</th>
<th>Late Decompensated Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Canine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>↓ (98°F–99°F)</td>
<td>↓↓ (96°F–98°F)</td>
<td>↓↓↓ (&lt;96°F)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>↑↑↑ (&gt;180 bpm)</td>
<td>↑ (&gt;150 bpm)</td>
<td>Normal to ↓ (&lt;140 bpm)</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>↑↑ (&gt;50 bpm)</td>
<td>↑ (&gt;50 bpm)</td>
<td>↑ to normal to agonal</td>
</tr>
<tr>
<td>Mentation</td>
<td>QAR</td>
<td>Obtunded</td>
<td>Obtunded to stupor</td>
</tr>
<tr>
<td>Mucous membrane color</td>
<td>Pale</td>
<td>Pale</td>
<td>Pale to muddy</td>
</tr>
<tr>
<td>Capillary refill time</td>
<td>&lt;1 sec</td>
<td>&lt;2 sec</td>
<td>≥2 sec</td>
</tr>
<tr>
<td>Mean arterial blood pressure</td>
<td>↓ to normal (70–80 mm Hg)</td>
<td>↓ (50–70 mm Hg)</td>
<td>↓↓ (&lt;60 mm Hg)</td>
</tr>
</tbody>
</table>

| **Feline**                  |                   |                           |                          |
| Temperature                 | ↓ (<97°F)         | ↓↓ (<95°F)                | ↓↓↓ (<90°F)              |
| Heart rate                  | ↑↑↑ (>240) or ↓ (160–180 bpm) | ↑↑ (>200 bpm) or ↓ (120–140 bpm) | ↑ (>180 bpm) or ↓↓ (≤120 bpm) |
| Respiratory rate            | ↑↑↑ (>60 bpm, open-mouth breathing) | ↑↑ (≥60 bpm)          | ↑ rate to agonal        |
| Mentation                   | QAR               | Obtunded                  | Obtunded to stupor       |
| Mucous membranes            | Pale              | Pale to white             | Variable (pale to white to muddy) |
| Capillary refill time       | <1 sec            | <2 sec                    | ≥2 sec                   |
| Systolic arterial blood pressure | ↓ to normal (80–90 mm Hg) | ↓ (50–80 mm Hg)        | ↓↓ (<50 mm Hg)          |

*Hypothetical values are given in parentheses to give the reader an idea of the approximate range of values found in each species at each stage of shock.*

QAR = quiet, alert, responsive

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¹Hypothetical values are given in parentheses to give the reader an idea of the approximate range of values found in each species at each stage of shock.  
QAR = quiet, alert, responsive
### Table 3. Physical Examination Findings During Shock States

<table>
<thead>
<tr>
<th>Physical Examination Finding</th>
<th>Compensated Shock</th>
<th>Acute Decompensated Shock</th>
<th>Late Decompensated Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Canine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rectal temperature</td>
<td>During vasoconstriction, peripheral vessels such as those in the GI tract are preferentially constricted. Reduced blood flow to the colon leads to a decreased rectal temperature.</td>
<td>Vasoconstrictive shunting of blood away from the GI tract continues. Simultaneously, the decreased cardiac output with worsening shock equates to less blood being delivered to the colon (and therefore a lower rectal temperature).</td>
<td>While vasoconstrictive shunting of blood is still in progress, the largest factor in the decreased temperature is the minimal amount of blood flow being delivered to the colon.</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Activation of β1-adrenergic receptors by epinephrine and norepinephrine leads to tachycardia.</td>
<td>As the myocardium receives less oxygen, the heart rate starts to decrease despite continued stimulation by the β1-adrenergic receptors.</td>
<td>Continued reduced myocardial perfusion and oxygen delivery leads to decreasing ability of the heart to contract and a reduced heart rate.</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>Multifactorial. Pain and stress lead to tachypnea. Respiratory rate also increases concurrent with perceived oxygen debt in the respiratory center in the brain.</td>
<td>As oxygen delivery decreases to the diaphragm and other muscles of respiration, the respiratory rate decreases.</td>
<td>Continued reduced oxygen delivery to the diaphragm and other muscles of respiration leads to worsening respiratory rate decline.</td>
</tr>
<tr>
<td>Mentation</td>
<td>Brain cells receive enough oxygen to function, but the patient is less responsive than normal. Pain also alters mentation.</td>
<td>As the cells of the brain receive less oxygen delivery, the patient's mentation declines (obtundation).</td>
<td>Continued reduced oxygen delivery to the brain leads to worsening obtundation.</td>
</tr>
<tr>
<td>Mucous membrane color</td>
<td>Pallor occurs primarily because of vasoconstriction and shunting of blood away from the periphery. Hyperemia develops because of massive release of vasodilatory mediators such as nitric oxide (seen in cases of sepsis and SIRS)</td>
<td>Pallor continues because of vasoconstriction and shunting of blood along with reduced ability to deliver blood to peripheral tissues.</td>
<td>Pallor occurs largely because of reduced peripheral perfusion. Muddy mucous membrane color develops when waste products of cellular metabolism diffuse into capillaries but are not removed from the region because of reduced perfusion.</td>
</tr>
<tr>
<td>Capillary refill time</td>
<td>Rapid refill caused by increased vascular tone (binding by epinephrine, norepinephrine, ANG II and vasopressin to receptors on vascular endothelium).</td>
<td>Blood vessels start to become refractory to constriction because of reduced oxygen delivery to endothelial cells. Vasodilatory effectors such as nitric oxide start to overwhelm vasoconstrictors.</td>
<td>Blood vessels lose the ability to constrict because of severely decreased oxygen delivery to those cells and the overwhelming influence of vasodilatory signals such as nitric oxide.</td>
</tr>
<tr>
<td>Mean arterial blood pressure</td>
<td>The body is able to maintain near normal pressure, primarily because of vasoconstriction.</td>
<td>Gradual loss of vascular tone occurs because of reduced oxygen delivery to the vascular endothelium.</td>
<td>Ability to vasoconstrict is lost because of reduced oxygen delivery; reduced cardiac output leads to reduced vascular volumes.</td>
</tr>
<tr>
<td><strong>Feline</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td>Same as canine.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td>Cats rarely display tachycardia during shock. Cats in shock are often bradycardic and become more so over time. Cats in compensated shock tend to have heart rates ≤160 bpm, and cats in decompensatory shock tend to have heart rates ≤100 bpm. It is not completely understood why cats respond to shock with relative to absolute bradycardia without first having a discernible period of tachycardia, despite having increases in SNS signals similar to dogs.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>Cats display profound tachypnea that can resemble respiratory distress. It is not completely understood why cats have such a dramatic response compared with dogs. However, the lungs are considered the “shock” organ in cats and, as such, receive markedly decreased perfusion during vasoconstrictive conditions, which may contribute to the extreme tachypnea and respiratory distress.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mentation</td>
<td>Same as canine.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mucous membranes/ capillary refill time</td>
<td>It is often difficult to visualize color and perform a capillary refill time even in healthy cats. However, it is likely that they follow a similar pattern to dogs.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean arterial blood pressure</td>
<td>Same as canine.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*SIRS = systemic inflammatory response syndrome; SNS = sympathetic nervous system; ANG II = angiotensin II*
increases sodium reabsorption in the distal convoluted tubule of the kidney. Water follows the sodium and is reabsorbed into the blood vessels, increasing blood volume. Whenever blood volume is increased, there is improved venous return (and therefore cardiac output).

**Antidiuretic Hormone**
Vasopressin has another effect in the body as antidiuretic hormone (ADH). Vasopressin and ADH are the same hormone; the two names reflect the two divergent effects in the body. When produced, ADH binds to V2 receptors in the collecting ducts of the kidney. This induces insertion of aquaporin channels into the collecting ducts to allow reabsorption of water from the ducts. ADH also stimulates thirst to increase the amount of water in the body and thereby improve blood volume and venous return.

**Clinical Signs Associated With Shock**
Building on the basic physiology of the shock response allows better understanding of the clinical signs associated with an animal presenting in shock. Patients go through three stages of shock: compensatory, acute decompensatory, and late decompensatory. Other terms for these stages are compensatory reversible shock, uncompensated reversible shock, and uncompensated irreversible shock. In the compensated stage, by virtue of the various physiologic mechanisms discussed above, the patient is able to maintain oxygen delivery to the tissues to preserve normal cellular metabolism. In the acute stage of decompensation, the demand for oxygen is greater than the delivery despite the action of the physiologic mechanisms; therefore, the cells are forced to switch to anaerobic metabolism, which yields less energy. In the later stages of decompensation, inappropriate oxygen delivery continues and the cellular demand for oxygen is not met, causing further anaerobic metabolism and less available ATP to the cells.

In veterinary medicine, determination of the patient’s stage of shock is based largely on the physical examination findings for that patient. See Table 2 for a summary of the physical examination findings found at each stage of shock and Table 3 for the reasons each finding exists at that stage.

The physical examination finding that has the most variability in cases of shock is the mucous membrane color. Based purely on the physiologic responses to which a patient is exposed during compensated shock, the expected mucous membrane color is pale because of vasoconstrictive shunting of blood away from the mucous membranes. However, in some cases, the mucous membranes can appear hyperemic. Hyperemic mucous membranes may be seen in diseases in which vasodilation overwhelms the vasoconstriction expected in compensated shock. Notable examples are septic shock and SIRS, in which vasodilatory mediators such as nitric oxide and cytokines that directly dilate the blood vessels are produced, leading to vasodilation. Later, in decompensatory shock, the pallor seen in SIRS and sepsis patients is caused by a lack of blood delivery to the mucous membranes, not vasoconstrictive mechanisms.

Cats may not display the classic sign of tachycardia seen in dogs. While there is no clear reason for this species difference, cats in shock that tend to display bradycardia (heart rate <140
bpm) or relative bradycardia (heart rate <160 bpm) are often septic or have SIRS.9,10

**Treatment**

In veterinary medicine, treatment for shock should be aimed at addressing the basic pathophysiologic mechanisms. Gauging a response to treatment for patients in shock is based on normalizing vital parameters and, often, peripheral blood pressure. There are very limited options available to clinicians to treat cases of shock (FIGURE 6).

Hypovolemic shock is primarily treated by large-volume fluid resuscitation. Crystalloid fluid doses for patients in shock are 90 mL/kg/h in dogs and 60 mL/kg/h in cats. It is recommended to give one-quarter to one-third of the calculated fluid dose to the animal in a bolus as quickly as possible and then reassess the patient’s vital parameters. The fluid bolus can be repeated as many times as necessary until the parameters have normalized or the hourly amount has been met. Further or additional steps might include administration of boluses of colloids (typically 5 to 10 mL/kg repeated until the patient is stabilized or to a maximum dose of 20 mL/kg for colloids such as hetastarch).

Obstructive shock is treated with fluids administered at shock doses in a vascular location where the fluids will return to the heart and not be trapped distal to the obstruction. For example, a patient with gastric dilatation-volvulus (GDV) should receive fluid in the cephalic veins, not the lateral saphenous veins. When applicable, the clinician should also attempt to relieve the obstruction (e.g., surgery to relieve GDV).

Cardiogenic shock does not involve decreased blood volume and instead is a failure of the heart to effectively pump blood to tissues. It is treated with positive inotropes (e.g., dobutamine) without fluid therapy. In some cases, drugs that cause vasodilation and reduce afterload, such as nitroprusside, are also used to improve cardiac output. It is important to limit or forgo fluid therapy in patients with cardiogenic shock because the heart may already be fluid overloaded by shunting of blood to the heart caused by vasocostriction during compensation.

Distributive shock is the most difficult form of shock to treat because it involves derangement of the microvasculature as well as the macrovasculature. These patients are treated with shock doses of fluids to improve hypovolemia resulting from increases in vascular permeability and maldistribution of fluids into the dilated vessels. Patients also require treatment with drugs to promote vasocostriction, such as vasopressin, dopamine, epinephrine, or norepinephrine and, in some cases, positive inotropes to improve myocardial depression (dobutamine). Finally, the underlying cause of the distributive shock must be addressed. Diseases leading to distributive shock may not only cause hypercoagulability (which can obstruct blood vessels) and release cytokines that cause depression of the myocardium but also, if untreated, prevent resolution of the patient’s condition. Also complicating the situation is the fact that improving macrovascular parameters such as heart rate or peripheral blood pressure does not necessarily mean that microcirculation (capillary perfusion) has been restored.4,6 However, at this time, clinicians do not have a clinically dependable bedside diagnostic test or tool that allows assessment of the microcirculatory response to resuscitation.

In any treatment situation, continuous reassessment of the patient’s vital parameters and status to determine whether resuscitation efforts have been successful is most important. If the patient does not seem to be improving as hoped, continue to administer treatment as suggested by the patient’s condition, but reassess the patient with a complete physical examination to look for indications of occult hemorrhage (e.g., into a body cavity or a fracture hematoma) that would lead to ongoing signs of hypovolemic shock. It is important to document that a refractory patient is not suffering from hypoglycemia caused by depleted liver stores occurring after exuberant cortisol release. Finally, especially in trauma patients, if the patient does not improve with resuscitation, imaging of body cavities is indicated to look further for blood loss or other abnormalities, such as pneumothorax, that might decrease venous return to the heart and further the shock condition.

Shock is a complex interaction between the inciting event and the body’s compensatory mechanisms. In understanding basic pathophysiology, clinicians should be able to better recognize patients in shock and to logically determine the best steps for resuscitation of these patients.

**References**

1. Which of the following lists the correct variables for cardiac output?
   a. heart rate, preload, stroke volume, oxygen saturation
   b. preload, afterload, contractility, heart rate
   c. hemoglobin, afterload, arterial oxygen content, stroke volume
   d. stroke volume, arterial oxygen content, hemoglobin, heart rate

2. Acute compensatory mechanisms that work to restore perfusion to the tissues demonstrate their effects within what time frame?
   a. days
   b. minutes
   c. hours
   d. months

3. Which of the following compensatory mechanisms does not happen within the first 30 minutes after onset of shock?
   a. release of aldosterone
   b. increase in heart rate
   c. increase in sympathetic tone
   d. release of norepinephrine

4. Which of the following is a stimulus for the release of catecholamines?
   a. hypernatremia
   b. hypertension
   c. hypoxemia
   d. hypercapnia

5. Which of the following is not a stimulus for the release of vasopressin?
   a. increased blood volume
   b. hypoxia
   c. nausea
   d. increased osmolarity

6. Which disease entity would most likely cause cardiogenic shock?
   a. hemoabdomen
   b. third-degree AV block
   c. pulmonary thromboembolism
   d. dilated cardiomyopathy

7. A dog with the following physical examination findings would be classified in which stage of shock?
   • Temperature: 94.5°F
   • Heart rate: 120 bpm
   • Respiratory rate: 60 breaths/min
   • Mentation: obtunded
   • Mucous membrane color: pale
   • Capillary refill time: >2 sec
   • Mean arterial pressure: 50 mm Hg
   a. Compensated
   b. Acute decompensated
   c. Acute compensated
   d. Late decompensated

8. Which of the following does not happen during distributive shock?
   a. decreased cardiac contractility
   b. embolization of small blood vessels
   c. increased contractility
   d. vasodilation

9. ________ is not generally associated with feline shock.
   a. Tachycardia
   b. Hypotension
   c. Hypothermia
   d. Tachypnea

10. ________ is a product of anaerobic cellular metabolism.
    a. Vasopressin
    b. Glucose
    c. Oxygen
    d. Lactate